Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults

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Time-series, cross-sectional, and prospective cohort studies have observed associations between mortality and particulate air pollution but have been limited by ecologic design or small number of subjects or study areas. The present study evaluates effects of particulate air pollution on mortality using data from a large cohort drawn from many study areas. We linked ambient air pollution data from 151 U.S. metropolitan areas in 1980 with individual risk factor on 552,138 adults who resided in these areas when enrolled in a prospective study in 1982. Deaths were ascertained through December, 1989. Exposure to sulfate and fine particulate air pollution, which is primarily from fossil fuel combustion, was estimated from national data bases. The relationships of air pollution to all-cause, lung cancer, and cardiopulmonary mortality was examined using multivariate analysis which controlled for smoking, education, and other risk factors. Although small compared with cigarette smoking, an association between mortality and particulate air pollution was observed. Adjusted relative risk ratios (and 95% confidence intervals) of all-cause mortality for the most polluted areas compared with the least polluted equaled 1.15 (1.09 to 1.22) and 1.17 (1.09 to 1.26) when using sulfate and fine particulate measures respectively. Particulate air pollution was associated with cardiopulmonary and lung cancer mortality but not with mortality due to other causes. Increased mortality is associated with sulfate and fine particulate air pollution at levels commonly found in U.S. cities. The increase in risk is not attributable to tobacco smoking, although other unmeasured correlates of pollution cannot be excluded with certainty. **Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath Jr CW. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 1995;151:669–74.**

Many studies have observed associations between particulate air pollution and human health (1). Increases in sickness and death associated with severe air pollution episodes have been well documented. Recent daily time-series studies have observed associations between daily mortality and changes in particulate air pollution (2–6) at levels below U.S. air quality standards. Elevated particulate air pollution has been associated with declines in lung function (6–9), increases in respiratory symptoms (6, 8–11), increases in respiratory hospitalizations (6, 12–13), and restricted activity (14, 15).

Ecologic cross-sectional studies have reported associations between mortality rates and sulfate or fine particulate pollution levels across metropolitan areas (16–19). Mortality risks of air pollution have also been estimated using data from a 14 to 16 year prospective follow-up of over 8,000 adults living in six U.S. cities (20) which controlled for individual differences in age, sex, cigarette smoking, and other factors. In both the ecologic studies and the recent prospective cohort study, mortality was more strongly associated with sulfate or fine particulate air pollution than with other measures of air pollution.

Particulate air pollution is a mixture of particles that vary in size, composition, and origin. Fine particles (those with aerodynamic diameters equal to or less than 2.5 μm) are the largest health concern because they can be breathed most deeply into the lung. This size range includes most sulfate particles (which generally make up the largest fraction of fine particles by mass). Unlike larger particles which are derived primarily from soil and other crustal materials, fine particles (including sulfates) are derived chiefly from combustion of fossil fuels in processes such as transportation, manufacturing, and power generation. Sulfate particles are commonly generated by conversion from primary sulfur emissions and a varying portion of sulfate particles may be acidic.

Previous studies of particulate pollution and mortality have been limited by ecologic design or small number of subjects or study areas. In the present study, a large cohort of adults living in 151 U.S. metropolitan areas was followed prospectively between 1982 and 1989. Ambient concentrations of sulfates and fine particles were used as indices of exposure to combustion source ambient particulate air pollution. Exposure to ambient air pollution was estimated from national data bases. Associations between

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mortality and particulate pollution were evaluated at pollution levels common to many U.S. metropolitan areas while directly adjusting for individual differences in smoking status, gender, age, education, and other risk factors.

METHODS

Study Population

This analysis relied on data for 552,138 men and women drawn from the American Cancer Society (ACS) Cancer Prevention Study II (CPS-II), an ongoing prospective mortality study of approximately 1.2 million adults (21). Participants were enrolled by ACS volunteers in the fall of 1982. They resided in all 50 states, the District of Columbia, and Puerto Rico, and were usually friends, neighbors, or acquaintances of the ACS volunteers. Enrollment was restricted to persons who were at least 30 yr of age and who were members of households with at least one individual 45 yr of age or more. Participants completed a confidential questionnaire which included questions about age, sex, weight, height, demographic characteristics, smoking history, alcohol use, occupational exposures, and other characteristics.

Vital status of participants was assessed from September 1, 1982 to December 31, 1989 using two approaches. First, vital status was determined by personal inquiries by the volunteers in September of 1984, 1986, and 1988. Second, automated linkage using the National Death Index (22) was used to extend vital status follow-up through December 31, 1989 and to identify deaths among the approximately 2% of participants who were lost to follow-up between 1982 and 1988. Death certificates were obtained for approximately 96% of deaths. A nosologist coded cause-of-death according to the International Classification of Diseases, 9th revision (ICD-9) (23), without knowledge of pollution levels. The analytic cohort used in this analysis included all CPS-II participants who provided complete questionnaire data on other risk factors evaluated, whose death certificates were obtained, and who resided in U.S. metropolitan areas within the 48 contiguous states (including the District of Columbia) that had available pollution data. Cohort characteristics are summarized in Table 1.

TABLE 1

SUMMARY CHARACTERISTICS OF SUBJECTS IN BASELINE ANALYTIC COHORT DERIVED FROM THE ACS, CPS-II STUDY COHORT, 1982–1989

Characteristics	Analysis with Sulfate Particles	Analysis with Fine Particles
Number of metropolitan areas	151	50
Number of subjects	552,138	295,223
Number of deaths	38,963	20,765
Age at enrollment, mean	56.5	56.6
Sex, % Female	56.0	55.9
Race, % White	94.2	94.0
Black	4.1	4.1
Other	1.7	1.9
Current cigarette smoker, %	22.0	21.6
Cigarettes/day, mean	22.0	22.1
Years smoked, mean	33.5	33.5
Former cigarette smoker, %	29.1	29.4
Cigarettes/day, mean	22.0	22.0
Years smoked, mean	22.3	22.2
Pipe/cigar smoker only, %	4.1	3.9
Passive smoke, hours/day, mean	3.2	3.2
Occupational exposure, %	20.0	19.5
Less than high school education, %	12.3	11.3
BMI, mean	25.1	25.0
Alcohol, drinks/day, mean	1.0	1.0
Sulfate particles, $\mu g/m^3$, mean (Standard deviation)	11.0 (3.6)	–
Sulfate particles, $\mu g/m^3$, range	3.6–23.5	–
Fine particles, $\mu g/m^3$, mean (Standard deviation)	–	18.2 (5.1)
Fine particles, $\mu g/m^3$, range	–	9.0–33.5

Air Pollution Exposure Estimates

Based on participant addresses at time of entry into the study and 3-digit zip code areas (24), each participant was assigned a metropolitan area of residence. Smoking status and other individual risk factors were assessed at the time of entry into the cohort. Pollution exposure also was assessed for a time period just prior to entry into the cohort.

Two indices of exposures to combustion source particulate air pollution were used. The first was mean concentration of sulfate air pollution for 1980 in the participant's area of residence based on data from the U.S. Environmental Protection Agency's (EPA) National Aerometric Data Base. Means were calculated as the average of annual arithmetic mean 24-h sulfate values for all monitoring sites in the Standard Metropolitan Statistical Areas or, in New England, New England County Metropolitan Areas that corresponded with defined areas of residence. Across the 151 metropolitan areas with matching data, mean sulfate concentrations averaged 11 $\mu g/m^3$ and ranged from 3.6 to 23.5 $\mu g/m^3$.

The second index of exposure to combustion source particulate air pollution was median fine particulate concentration for 1979 to 1983 calculated from the EPA dichotomous sampler network by Lipfert and coworkers in a population-based cross-sectional analysis of mortality across U.S. cities (17). There were 50 metropolitan areas with matching data that could be analyzed using this pollution measure. Across these 50 areas, median fine particulate concentrations averaged 18.2 $\mu g/m^3$ and ranged from 9.0 to 33.5 $\mu g/m^3$.

Because both fine and sulfate particles are derived chiefly from the combustion of fossil fuels and because sulfates make up the largest fraction of fine particles by mass, both pollution measures serve as indexes of combustion source particulate pollution and are highly correlated. For the 47 metropolitan areas with both pollution measures, the Pearson correlation coefficient between sulfate and fine particulate pollution was 0.73 ($p < 0.001$).

Statistical Analysis

Adjusted mortality relative risk ratios were estimated using multiple regression analysis based on the Cox proportional hazards model (25) using SAS/STAT Software (26). The time variable used in the model was survival time from date of enrollment. Survival times of participants who did not die were censored at the end of the study period. Adjusted risk ratios were calculated and reported for differences in air pollution equal to the range of pollution observed across the areas (Table 1). All models were stratified by 5-yr age categories, gender, and race (white, black, and other) which allowed each sex-race-age category to have its own baseline hazard. Models were estimated including air pollution as an independent variable. To control for smoking at entry, the following variables were included in the models: an indicator variable for current smoker, an indicator variable for pipe and/or cigar smoker only, years smoked for current smoker, cigarettes per day for current smoker, years smoked for former smoker, number of cigarettes per day for former smoker, and number of hours per day exposed to passive cigarette smoke. To control for other individual risk factors, several other variables were included: body-mass index (BMI), drinks per day of alcohol, a variable indicating less than a high school education, and a variable indicating regular occupational exposure to any of the following: asbestos, chemicals/acids/solvents, coal or stone dusts, coal tar/pitch/asphalt, diesel engine exhaust, or formaldehyde.

Cox proportional hazards models were estimated separately for three cause-of-death categories: lung cancer (ICD-9 162), cardiopulmonary disease (ICD-9 401–440 and 460–519), and all others. Deaths not in that specific category were censored at time of death. To evaluate the robustness of the estimated effects, the models were reestimated after separating the data by smoking status, and gender. Additionally, to evaluate if the results were confounded by differences in climates across the metropolitan areas, weather variables that accounted for relatively hot or cold conditions were added to the models.

Ecologic Analysis

To compare these results with more commonly available population based mortality rates, U.S. metropolitan area mortality rates for 1980 were obtained from the National Center for Health Statistics (27). These population-based mortality rates were from metropolitan areas that correspond approximately

to areas used in this study. These mortality rates were adjusted based on age-sex-race specific population counts from the 1980 census (28) (with seven age categories and a white/nonwhite race designation). The adjusted mortality rates were then correlated with sulfate and fine particulate pollution levels.

RESULTS

Adjusted Mortality Risk

Although small relative to active smoking (Table 2), an association between mortality and air pollution was observed. The latter association persisted after adjusting for age, sex, race, cigarette smoking, pipe and cigar smoking, exposure to passive cigarette smoke, occupational exposure, education, BMI, and alcohol use. For all-cause, cardiopulmonary, and lung cancer mortality, the associations with sulfates were statistically significant ($p < 0.001$). For all-cause and cardiopulmonary mortality, significant associations were also found using fine particulate matter as the index of air pollution. Mortality due to other causes was not significantly associated with pollution levels (Table 2).

Lung cancer mortality was associated with combustion source air pollution when sulfates were used as the index but not when fine particles were used as the index. To evaluate whether this inconsistency was due to the use of different study areas or different pollution measures, sulfate pollution measures were included in models that were restricted to use data only from the 47 metropolitan areas that had both sulfate and fine particulate measures. The adjusted mortality risk ratios (and 95% CI) for lung cancer and cardiopulmonary disease mortality for all persons combined controlling for the other risk factors were 1.44 (1.11 to 1.86) and 1.20 (1.08 to 1.34), respectively. The results were similar to those from our initial analysis suggesting that the inconsistency was not due to differences in study areas, but lung cancer seems to be more strongly associated with sulfate particles than the more general index of fine particulate mass.

The association between air pollution and all-cause and cardiopulmonary mortality was consistent across both men and women, and among smokers and nonsmokers. Cox proportional hazard regression models showed no statistically significant differences in pollution-related mortality risk when the data were separated by smoking and gender strata (Table 3). Estimated pollution-related mortality risk was as high for never-smokers as it was for ever-smokers and as high for women as it was for men.

After adjusting for cigarette smoking, the association between air pollution and all-cause and cardiopulmonary mortality was not sensitive to the inclusion of BMI, alcohol consumption, education, and occupational exposure variables. There was also little evidence that the results were due to differences in climates across the metropolitan areas. Normal daily high, low, or mean temperature was not correlated with either sulfate or fine particulate pollution. Absolute Pearson correlation coefficients between mean temperature variables and sulfate and fine particulate pollution

TABLE 2

ADJUSTED MORTALITY RISK RATIOS (AND 95% CONFIDENCE INTERVALS) BY CAUSE OF DEATH FOR CIGARETTE SMOKING AND FOR A DIFFERENCE IN POLLUTION*

Cause of Death	Current Smoker†	Sulfates‡ (19.9 μg/m³)	Fine Particles‡ (24.5 μg/m³)
All	2.07 (1.75–2.43)	1.15 (1.09–1.22)	1.17 (1.09–1.26)
Lung cancer	9.73 (5.96–15.9)	1.36 (1.11–1.66)	1.03 (0.80–1.33)
Cardiopulmonary	2.28 (1.79–2.91)	1.26 (1.16–1.37)	1.31 (1.17–1.46)
All other	1.54 (1.19–1.99)	1.01 (0.92–1.11)	1.07 (0.92–1.24)

* Difference in pollution equal to the most polluted areas compared with the least polluted using sulfates and fine particles as measures of combustion source air pollution.

† Risk ratios for cigarette smoking are estimated from the model using sulfate data and correspond to the risk of death for a current smoker with 25 yr of smoking 20 cigarettes per day as compared with a never-smoker. Risk ratios have been adjusted for age, sex, race, exposure to passive cigarette smoke, body-mass index, drinks per day of alcohol, education, and occupational exposure.

‡ Risk ratios have been adjusted for age, sex, race, cigarette smoking, exposure to passive cigarette smoke, body-mass index, drinks per day of alcohol, education, and occupational exposure.

TABLE 3

ADJUSTED MORTALITY RISK RATIOS* (AND 95% CI) FOR THE MOST POLLUTED AREAS COMPARED WITH THE LEAST POLLUTED FOR ALL-CAUSE AND CARDIOPULMONARY DEATHS SEPARATED BY GENDER AND SMOKING STATUS

	Sulfates (19.9 μg/m³)			Fine Particles (24.5 μg/m³)		
	All Cause	Lung Cancer	Cardiopulmonary	All Cause	Lung Cancer	Cardiopulmonary
All combined	1.15 (1.09–1.22)	1.36 (1.11–1.66)	1.26 (1.16–1.37)	1.17 (1.09–1.26)	1.03 (0.80–1.33)	1.31 (1.17–1.46)
Women	1.18 (1.06–1.30)	1.17 (0.80–1.72)	1.39 (1.20–1.61)	1.16 (1.02–1.32)	0.90 (0.56–1.44)	1.45 (1.20–1.78)
Men	1.14 (1.06–1.23)	1.43 (1.13–1.81)	1.20 (1.08–1.33)	1.18 (1.07–1.30)	1.10 (0.81–1.47)	1.24 (1.08–1.41)
Never-smokers	1.18 (1.06–1.30)	1.51 (0.73–3.11)	1.36 (1.19–1.58)	1.22 (1.07–1.39)	0.59 (0.23–1.52)	1.43 (1.18–1.72)
Women	1.20 (1.06–1.36)	1.61 (0.66–3.92)	1.44 (1.20–1.74)	1.21 (1.02–1.43)	0.65 (0.21–2.06)	1.57 (1.23–2.01)
Men	1.14 (0.97–1.34)	1.36 (0.40–4.66)	1.28 (1.03–1.58)	1.24 (1.00–1.54)	0.49 (0.09–2.66)	1.24 (0.93–1.67)
Ever-smokers	1.14 (1.06–1.23)	1.35 (1.10–1.66)	1.20 (1.08–1.33)	1.15 (1.05–1.26)	1.07 (0.82–1.39)	1.24 (1.08–1.42)
Women	1.14 (0.97–1.33)	1.10 (0.72–1.68)	1.30 (1.01–1.66)	1.10 (0.90–1.33)	0.95 (0.57–1.58)	1.27 (0.92–1.74)
Men	1.14 (1.05–1.24)	1.44 (1.14–1.83)	1.17 (1.05–1.32)	1.16 (1.05–1.29)	1.12 (0.83–1.52)	1.23 (1.06–1.43)

* Risk ratios have been adjusted for age, sex, race, cigarette smoking, exposure to passive cigarette smoke, body-mass index, drinks per day of alcohol, education, and occupational exposure.

were all less than 0.1 and statistically insignificant ($p > 0.25$). However, on average sulfate particulate levels were slightly lower in both the relatively cold and relatively hot metropolitan areas. Therefore indicator variables were created for the relatively hot and cold cities (those with normal mean temperatures greater than 60° F and less than 50° F). The inclusion of these weather indicator variables in the Cox proportional hazard models had little impact on the estimated association between particulate air pollution and mortality. When these weather indicator variables were included in the models, adjusted relative risk ratios (and 95% confidence intervals) for lung cancer and cardiopulmonary mortality equaled 1.36 (1.11 to 1.66) and 1.23 (1.13 to 1.34) respectively when sulfate is used as the pollution measure and 1.05 (0.82 to 1.36) and 1.26 (1.13 to 1.40) respectively when fine particulate pollution is used as the pollution measure.

Ecologic Comparison

Age-, sex-, and race-adjusted population-based mortality rates for 1980 (using metropolitan areas also used in this prospective cohort study) are plotted against sulfates and fine particles in Figures 1 and 2, respectively. Sulfate and fine particle pollution were associated with higher mortality rates. Regression coefficients between mortality rates and air pollution equaled 10.5 (SE = 1.3) and 8.0 (SE = 1.4) deaths/year/100,000 persons in the population per $\mu g/m^3$ of sulfate and fine particulate pollution respectively. Although this ecologic analysis did not control for risk factors except age, sex, and race, these correlations were statistically significant ($p < 0.001$) and demonstrated an association similar to that observed in the prospective cohort study of participants from the same communities. Using the mean age-sex-race adjusted mortality rate as the baseline risk, estimated risk ratios for the most polluted city versus the least polluted city using sulfate and fine particulate measures of pollution equaled 1.25 and 1.24, respectively.

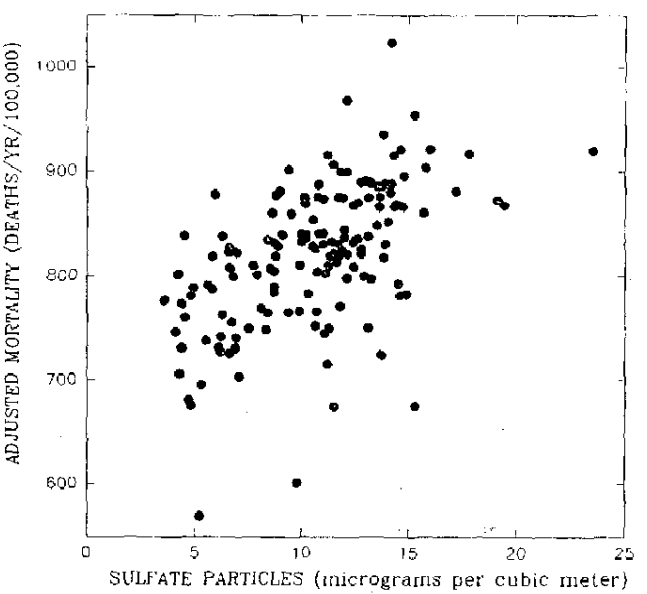

Figure 1. Age-, sex-, and race-adjusted population-based mortality rates for 1980 plotted against mean sulfate air pollution levels for 1980. Data from metropolitan areas that correspond approximately to areas used in prospective cohort analysis.

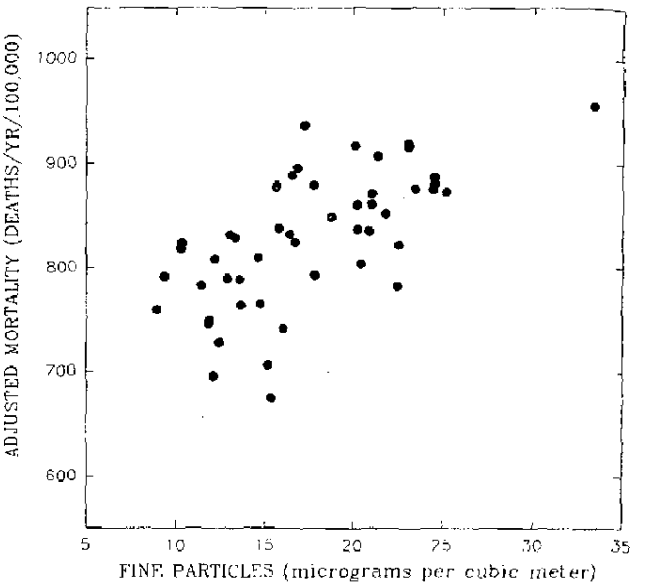

Figure 2. Age-, sex-, and race-adjusted population-based mortality rates for 1980 plotted against mean fine particulate air pollution levels for 1979 to 1983. Data from metropolitan areas that correspond approximately to areas used in prospective cohort analysis.

DISCUSSION

In this study, sulfate and fine particulate air pollution were associated with a difference of approximately 15 to 17% between mortality risks in the most polluted cities and those in the least polluted cites. Previous studies have observed similar results but have been limited by ecologic design or by small number of subjects or study areas. This study differs fundamentally from purely ecologic cross-sectional studies in using a prospective cohort design that allows for direct control of other individual risk factors, particularly cigarette smoking. Furthermore, because this study linked ambient air pollution data from national data bases with a large nationwide prospective cohort, this study is larger and represents a wider geographic area.

Although the increased risk associated with air pollution was small compared with that from cigarette smoking, results of this study suggest that the association between pollution and mortality was not likely due to inadequate control of smoking: (1) The associations between air pollution and mortality persisted after controlling for cigarette smoking status, pipe and/or cigar smoking, years smoked, and cigarettes smoked per day for both current and former smokers, and hours per day exposed to passive cigarette smoke. (2) Associations between particulate air pollution and mortality were as large and statistically significant for never-smokers as they were for ever-smokers.

Other potential sources of confounding are inadequate control of occupational, socioeconomic, or weather factors. Nevertheless, such residual confounding seems unlikely because: (1) The association between pollution and mortality was not very sensitive to the inclusion of variables reflecting occupational exposure, education, BMI, alcohol consumption, and relatively hot or cold weather conditions. (2) In the U.S., men are more likely to be employed in jobs with high industrial exposure to dust and fumes than women; yet the association between mortality and particulate air pollution was as high for women as for men. (3) Associations between particulate air pollution and mortality have also been

observed in daily time-series studies from various cities (2–6), yet community-specific occupational and socioeconomic conditions do not fluctuate daily with pollution levels.

In this study, individual data on smoking and other risk factors were obtained directly by questionnaire. Although accurate measures of lifetime personal exposure to air pollution would be ideal for many research purposes, such measures are unavailable and impractical for large cohorts. Furthermore public policy and pollution abatement strategies typically (and often necessarily) focus on ambient concentrations of air pollutants. Therefore, exposures to air pollution were estimated using ambient air pollution for metropolitan areas based on existing air pollution monitoring data.

The pollution data characterize differences in exposure between metropolitan areas for a specific period of time that corresponds roughly to the period of cohort enrollment and to the period when EPA dichotomous sampler network data were available. The biologically relevant exposure window for at least some of the mortality outcomes under study includes time periods for up to 15 or more years prior to death. The lack of long-term exposure data, therefore, results in some misclassification of exposure, the magnitude of which is largely dependent on the temporal constancy of the absolute and relative levels of pollution. Data from six cities in the East and Midwest U.S., indicate that annual average fine and sulfate particulate concentrations were relatively constant from the mid-1970s through the mid-1980s (20), suggesting that the pollution data used in this analysis also partially serve in proxy for longer-term exposures. While the lack of long-term exposure data constrains our ability to differentiate the time dependency of exposure and mortality, the air pollution measures used in this study partially reflect exposure to air pollution for periods preceding enrollment into the cohort. Furthermore, related exposure misclassification is unlikely to result in spurious associations between pollution and mortality. To the extent that the available exposure data do not adequately represent long-term exposure, the total chronic effects of air pollution may be underestimated.

Sulfate and fine particulate pollution data for a large number of communities are only available from central site ambient air pollution monitoring networks. These data can estimate variability in pollution exposure between communities, but within-community spatial variability of sulfate or fine particulate concentrations cannot be estimated for most of the areas included in this study. However, long-term transport and large-scale mixing of combustion products result in concentrations of sulfates and fine particles that are relatively uniform within communities (29). Variability of exposure within communities can also be due to differences in indoor versus outdoor concentrations and differences in time spent outdoors. Studies that conducted detailed monitoring within selected communities have concluded that measured indoor and personal exposures to sulfate and fine particles are strongly correlated with and similar to measured outdoor concentrations (30–32). Furthermore, these studies observed little within-community spatial variation in outdoor sulfate or fine particulate concentrations compared with between-community variations. For example, in Uniontown, Pennsylvania (31), nearly all of the variability in outdoor home site concentrations of sulfate particles was explained by concentrations at the central stationary ambient monitoring site (R^2 = 0.92); fine particle concentrations throughout Riverside, California (32) were similarly well estimated from the stationary central site monitor.

This study was limited by the use of death certificates to identify causes of death. Studies that used antemortem evidence or autopsy reports to verify cause of death have found that deaths due to respiratory disease are often recorded on the death certificate as cardiovascular (or circulatory) disease (33–35). Given this cross-coding between pulmonary and cardiovascular deaths and the potential that cross-coding may vary with age, survival analysis controlling for age and conducted separately for cardiovascular and pulmonary disease deaths may result in unstable and potentially biased estimates of pollution-related mortality risks. To avoid these problems, cardiovascular and pulmonary deaths were combined. All-cause mortality, or cardiovascular and pulmonary disease mortality grouped together, were consistently associated with air pollution.

This study and related epidemiologic studies provide little information on specific biologic mechanisms responsible for the observed effects. Additional research that will help provide a toxicologic framework for interpreting these findings is needed. Nevertheless, the biologic plausibility of these results is enhanced by several observations: (*1*) The increase in all-cause mortality associated with air pollution observed in this prospective cohort study is consistent with ecologic correlations presented here for the same metropolitan areas and with associations observed in several previous population-based cross-sectional mortality studies (16–19). (*2*) The results of this study are similar to those of the Harvard Six-Cities prospective cohort study (20) which estimated that the relative risk of mortality was 26% higher in the most, compared with the least polluted city. (*3*) Acute exposure studies have observed that particulate air pollution levels common to many of the metropolitan areas included in this study are associated with declines in lung function (6–9), increases in respiratory symptoms (6, 8, 9), increases in respiratory hospitalizations (6, 12, 13), restricted activity due to respiratory illness (14, 15), and increased mortality, especially respiratory and cardiovascular mortality (2–6). (*4*) While this and related epidemiologic studies suggest that combustion source air pollution is associated with a coherent cascade of cardiopulmonary health effects, this pollution is not typically associated with noncardiopulmonary health endpoints.

Findings of this study suggest that the associations observed between particulate air pollution and mortality in U.S. communities are not due to confounding by other risk factors, especially cigarette smoking. In combination with daily time-series mortality and morbidity studies, they suggest that combustion source air pollutants may be important contributing factors causing respiratory illness and early mortality due to cardiopulmonary diseases.

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